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Targeted Cancer Therapy: Pharmacological Advances in Precision Oncology

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ABSTRACT
Cancer remains one of the major causes of morbidity and mortality worldwide and it creates a continuous need for more effective and personalized treatment strategies. Targeted cancer therapy has changed the field of oncology by focusing on specific molecular abnormalities involved in tumor growth, survival, and progression. By selectively interfering with cancer-related molecular pathways, targeted therapies can improve treatment precision and reduce unnecessary damage to normal tissues. Precision oncology brings together molecular diagnostics, pharmacogenomics, biomarker testing, and companion diagnostics to support individualized treatment selection and improve clinical outcomes. This narrative review summarizes recent pharmacological advances in targeted cancer therapy and examines their role in precision oncology. A structured literature search was conducted using major scientific databases and authoritative sources, with emphasis on peer-reviewed articles, clinical studies, and international guidance related to targeted therapies, molecular biomarkers, pharmacogenomics, and precision medicine. The review covers the molecular basis of precision oncology, major classes of targeted anticancer agents, predictive biomarkers, companion diagnostics, and clinical applications across different cancer types. Mechanisms responsible for drug resistance and emerging therapeutic strategies are also discussed. Recent developments in antibody-drug conjugates, liquid biopsy, multi-omics technologies, and artificial intelligence are highlighted for their potential contribution to more personalized cancer care. Several challenges continue to affect the clinical use of targeted therapies like drug resistance, tumor heterogeneity, limited access to molecular testing, and the high cost of treatment. Additionally, progress in fields of molecular pharmacology, biomarker-guided treatment, and innovative therapeutic strategies may expand the clinical applications of precision oncology. Overall, targeted cancer therapy gradually strengthens personalized cancer management and remains an important area for future oncology research and clinical practice.
Keywords: Molecular biomarkers, Pharmacogenomics, Precision medicine, Precision oncology, Targeted cancer therapy.

Introduction

Cancer remains one of the leading causes of illness and death worldwide even after major advances in prevention, diagnosis, and treatment. Survival for patients is improved by many conventional treatment strategies like surgery, chemotherapy, and radiotherapy. Chemotherapy is the main systemic treatment for several decades, but its lack of selectivity leads to damage of healthy tissues along with cancer cells. This non-specific action contributes to adverse effects, treatment-related toxicity, and variable therapeutic outcomes in patients [1].

A better understanding of cancer biology has transformed the way many malignancies are treated. Research has shown that cancer develops through the accumulation of genetic and molecular alterations that disrupt normal cellular processes such as growth, proliferation, apoptosis, angiogenesis, and DNA repair. These molecular changes differ in each patient, even within the same type of cancer. This results in significant variation in disease progression and treatment response [2]. This knowledge has shifted cancer management toward precision oncology, where treatment decisions are guided by the molecular characteristics of an individual's tumor instead of depending only on the tissue or organ of origin.

Precision oncology combines genomic profiling, molecular diagnostics, pharmacogenomics, and targeted therapeutics to identify the most appropriate treatment for each patient. Advances in next-generation sequencing (NGS), liquid biopsy, transcriptomics, proteomics, and other high-throughput technologies have made it possible to detect clinically relevant molecular alterations with greater accuracy and speed. These technologies have expanded the identification of biomarkers and supported more personalized therapeutic strategies in a wide range of cancers [3,4].

Targeted cancer therapy is one of the most important components of precision oncology. They produce an action on specific molecular abnormalities that cause tumor development and progression. These drugs interfere with signalling pathways or cellular processes which are essential for cancer cell survival while minimizing damage to normal tissues. Several classes of

targeted agents are now available, including tyrosine kinase inhibitors, monoclonal antibodies, antibody-drug conjugates, poly (ADP-ribose) polymerase (PARP) inhibitors, cyclin-dependent kinase (CDK) 4/6 inhibitors, BRAF and MEK inhibitors, and phosphoinositide 3-kinase (PI3K) inhibitors. Many of these therapies have produced meaningful progresses in clinical outcomes for patients with breast cancer, non-small cell lung cancer, colorectal cancer, melanoma, leukaemia, ovarian cancer, and several other malignancies [5,6].

The success of targeted therapy depends on the identification of predictive biomarkers that indicate whether a patient may respond to a specific treatment. Molecular alterations such as mutations, gene amplifications, chromosomal rearrangements, and abnormal protein expression act as biomarkers for treatment selection. Companion diagnostic tests have also become an essential part of precision oncology. These tests assist in identifying patients who probably may benefit from targeted agents while avoiding ineffective therapies. Pharmacogenomic information also supports individualized treatment by improving drug selection, dose optimization, and safety monitoring [2,7].

Remarkable progress has been made in targeted therapies but several challenges continue to limit the long-term success of targeted cancer therapy. Primary and acquired drug resistance are major causes of treatment failure. Tumor heterogeneity, clonal evolution, activation of alternative signalling pathways, and changes in the tumor microenvironment can reduce treatment effectiveness over time. High treatment costs, limited access to genomic testing, unequal availability of precision medicine services, and the need for continuous biomarker validation also influence the successful implementation of precision oncology in routine clinical practice [4,8].

Recent pharmacological advances are gradually expanding the scope of precision oncology. The development of next-generation kinase inhibitors, bispecific antibodies, antibody-drug conjugates, targeted protein degraders, radioligand therapy, and combination treatment strategies have created new opportunities to improve therapeutic outcomes. Integration of artificial intelligence with genomic analysis and clinical decision support is also strengthening treatment planning and supporting the continuous evolution of precision oncology [3,6]. This narrative review summarizes the pharmacological advances that have shaped targeted cancer therapy within the framework of precision oncology. It discusses the molecular basis of targeted treatment, major drug classes, biomarkers and companion diagnostics, and clinical applications across different cancer types. It also examines mechanisms of drug resistance, emerging therapeutic strategies, and future directions that may further improve personalized cancer care. The evolution of cancer treatment has progressed from conventional chemotherapy to highly selective targeted therapies supported by molecular diagnostics and precision oncology.

Fig 1 illustrates the major milestones in this transition and highlights the shift toward personalized cancer treatment. The advances summarized in Fig 1 have established the foundation for precision oncology and have accelerated the development of targeted therapies based on specific molecular modifications. The following section discusses the molecular basis of precision oncology and the pharmacological principles underlying targeted cancer therapy.

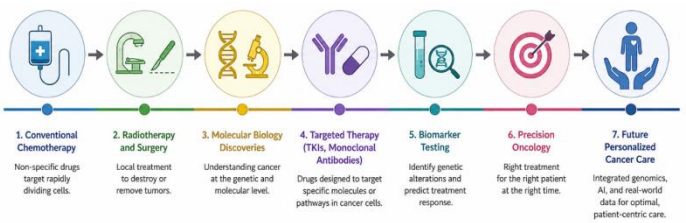
1. Conventional Chemotherapy
Non-specific drugs target rapidly dividing cells.
2. Radiotherapy and Surgery
Local treatment to destroy or remove tumors.
3. Molecular Biology Discoveries
Understanding cancer at the genetic and molecular level.
4. Targeted Therapy (TKIs, Monoclonal Antibodies)
Drugs designed to target specific molecules or pathways in cancer cells.
5. Biomarker Testing
Identify genetic alterations and predict treatment response.
6. Precision Oncology
Right treatment for the right patient at the right time.
7. Future Personalized Cancer Care
Integrated genomics, AI, and real-world data for optimal, patient-centric care.

Fig 1 Evolution of Cancer Therapy: From Conventional Chemotherapy to Precision Oncology.

Literature Search Strategy

A narrative literature review was conducted to summarize recent pharmacological advances in targeted cancer therapy and their role in precision oncology. Electronic databases, including PubMed, Scopus, Web of Science, Google Scholar, and ClinicalTrials.gov, were searched to identify relevant publications. The literature search covered studies published between January 2015 and June 2026. Landmark studies published before 2015 were also considered when they provided important historical or scientific background on targeted cancer therapy and precision oncology.

The search strategy combined Medical Subject Headings (MeSH) and free-text keywords. The main search terms included "targeted cancer therapy," "precision oncology," "precision medicine," "molecular targeted therapy," "pharmacogenomics," "companion diagnostics," "biomarkers," "tyrosine kinase inhibitors," "monoclonal antibodies," "antibody-drug conjugates," "PARP inhibitors," "CDK4/6 inhibitors," "BRAF inhibitors," "KRAS inhibitors," "PI3K inhibitors," "radioligand therapy," "protein degraders," "drug resistance," and "personalized cancer treatment." Different combinations of these keywords were used to retrieve the most relevant publications.

Eligibility Criteria

The review included original research articles, randomized clinical trials, observational studies, systematic reviews, narrative reviews, meta-analyses, clinical practice guidelines, regulatory documents, and landmark publications related to targeted cancer therapy and precision oncology. Publications describing molecular mechanisms, pharmacological characteristics, biomarker-guided treatment, companion diagnostics, resistance mechanisms, and emerging targeted therapies were considered for inclusion. Articles were excluded if they were unrelated to precision oncology, focused only on conventional chemotherapy without discussing targeted therapies, lacked sufficient scientific information, were duplicate publications, conference abstracts without full-text availability, editorials, letters to the editor, or were published in languages other than English.

Study Selection and Data Collection

Titles and abstracts identified through the database search were screened to determine their relevance to the objectives of this review. Full-text articles were then evaluated for eligibility. Publications that provided comprehensive information on molecular targets, targeted drug classes, clinical applications,

pharmacogenomics, biomarkers, companion diagnostics, therapeutic resistance, and recent pharmacological developments were selected for detailed review. Relevant information from each publication was extracted and organized according to predefined themes. Data collected included categories like type of targeted therapy, molecular target, mechanism of action, approved clinical indications, predictive biomarkers, therapeutic benefits, limitations, adverse effects, resistance mechanisms, and future developments. Recent regulatory approvals from agencies such as the U.S. Food and Drug Administration (FDA) and the European Medicines Agency (EMA) were also reviewed.

Data Synthesis

The selected literature was analysed using a qualitative narrative approach. Findings from different studies were grouped into major thematic areas, including the evolution of precision oncology, molecular basis of targeted therapy, pharmacology of targeted anticancer drugs, biomarker-guided treatment selection, clinical applications across different cancer types, mechanisms of therapeutic resistance, and emerging pharmacological advances. Information from multiple sources was compared and integrated to provide a broad overview of current knowledge in precision oncology. Recent developments and remaining challenges in the field were also identified

Reporting

The literature search and study selection process were performed according to the general principles of the PRISMA 2020 reporting framework to improve transparency and reproducibility. quantitative meta-analysis, statistical pooling, and formal assessment of study quality or risk of bias were not performed as it is a narrative review.

Evolution of Cancer Therapy: From Chemotherapy to Precision Oncology

Cancer treatment has changed remarkably since the past few decades. Surgery, radiotherapy, and conventional chemotherapy have been the primary treatment options for many malignancies. Chemotherapy remains an important component of cancer management because it destroys rapidly dividing cells and can reduce tumor burden in both localized and advanced disease. Its non-selective mechanism of action also affects healthy tissues, resulting in adverse effects such as myelosuppression, gastrointestinal toxicity, alopecia, and fatigue. Differences in tumor biology among patients also contribute to variations in treatment response and clinical outcomes. This highlights the need for more individualized therapeutic strategies [1,12].

Advances in cancer biology have transformed the understanding of tumor development. Cancer is now recognized as a molecular disease caused by genetic and epigenetic alterations. it disrupts normal cellular processes like cell proliferation, apoptosis, angiogenesis, DNA repair, and intracellular signalling. These molecular abnormalities differ among patients and even among tumors originating from the same tissue. This biological diversity has shifted the focus of cancer treatment from a disease-centered model to one based on the molecular characteristics of each patient's tumor [2,12].

The development of genomic technologies has enhanced the growth of precision oncology. High-throughput techniques such as next-generation sequencing (NGS) can identify mutations, gene amplifications, chromosomal rearrangements, copy number variations, and other genomic changes associated with cancer. These advances have improved the identification of actionable molecular targets and have strengthened personalized treatment design by matching patients with therapies directed against specific genetic abnormalities [2–4].

Precision oncology combines genomic profiling, molecular diagnostics, pharmacogenomics, and clinical information to guide individualized treatment decisions. The objective is to select therapies that are most appropriate for the molecular profile of each patient's tumor while reducing unnecessary toxicity and avoiding ineffective treatments. Continuous improvements in bioinformatics and genomic data analysis have further supported the integration of molecular testing into routine oncology practice [2–4,8].

Targeted cancer therapy has become one of the most important therapeutic strategies in precision oncology. conventional cytotoxic drugs produce generalized action whereas, targeted agents interact with specific molecular targets that regulate tumor growth, survival, angiogenesis, invasion, and metastasis. These therapies act on abnormal proteins or signalling pathways that lead to malignant progression and have improved treatment specificity for many cancers. Protein kinase inhibitors and monoclonal antibodies represent major classes of targeted agents currently used in clinical practice [5,6,13].

A major breakthrough in targeted therapy was the introduction of imatinib for chronic myeloid leukaemia. By selectively inhibiting the BCR-ABL tyrosine kinase produced by the Philadelphia chromosome, imatinib dramatically improved survival and changed the standard of care for patients with chronic myeloid leukaemia. Its success demonstrated that identifying and inhibiting a disease-driving molecular target can provide much clinical benefit and encouraged the development of many additional targeted therapies [5].

Since then, numerous targeted agents have entered clinical practice. Epidermal growth factor receptor (EGFR) inhibitors have improved outcomes in patients with EGFR-mutated non-small cell lung cancer, while HER2-targeted therapies have significantly increased survival in HER2-positive breast and gastric cancers. Targeted inhibition of BRAF has transformed the treatment of BRAF-mutated melanoma, and selective KRAS G12C inhibitors have expanded treatment options for patients with specific molecular subtypes of lung cancer. These advances demonstrate the continuous progress of precision oncology and the growing importance of biomarker-guided therapy [13–16].

The effectiveness of targeted therapy depends on precise identification of predictive biomarkers. Molecular testing and companion diagnostic assays identify patients who are expected to benefit from specific targeted agents. Biomarker-guided treatment selection improves therapeutic precision, supports individualized care, and reduces unnecessary exposure to ineffective therapies. Additionally, the increasing availability of complete genomic profiling has strengthened precision oncology by allowing simultaneous evaluation of multiple clinically relevant genetic alterations [4,7,8,10,11].

Several challenges continue to influence the long-term success of targeted cancer therapy even after significant progress. Primary and acquired resistance is identified as one of the major causes of treatment failure. Secondary mutations, activation of alternative signalling pathways, tumor heterogeneity, clonal evolution, and interactions with the tumor microenvironment can reduce treatment effectiveness during disease progression. Continuous research is focused on identifying resistance mechanisms and developing new therapeutic strategies to overcome them [4,17].

Recent advances in multi-omics technologies have expanded the scope of precision oncology beyond genomic analysis alone. Integration of genomic, transcriptomic, proteomic, metabolomic, and epigenomic data provides a more complete understanding of tumor biology. to analyse these complex datasets, identify clinically relevant patterns, and support treatment selection Artificial intelligence and advanced computational methods are increasingly used. These developments continue to improve patient categorization and accelerate the discovery of new therapeutic targets for precision oncology [2–4].

The transition from conventional chemotherapy to biomarker-guided targeted therapy represents one of the most significant achievements in modern oncology. Precision oncology continues to reshape cancer management by integrating molecular diagnostics, pharmacogenomics, and targeted therapeutics into routine clinical practice. Ongoing pharmacological research and persistent advances in molecular medicine are expected to expand treatment options and also improve outcomes for patients with different types of cancer [2,6,8].

Molecular Basis of Precision Oncology

Precision oncology is based on the principle that cancer develops through the accumulation of molecular alterations that disrupt normal cellular functions. These alterations affect genes involved in cell growth, survival, differentiation, DNA repair, angiogenesis, and immune regulation. Since each tumor possesses a unique molecular profile, identifying these abnormalities helps clinicians to select therapies that specifically target the biological mechanisms responsible for tumor progression [2,12].

Cancer is occurred by a combination of inherited and acquired genetic changes. Somatic mutations are the most common alterations detected in solid tumors and involve oncogenes, tumor suppressor genes, and DNA repair genes. Activation of oncogenes such as EGFR, HER2 (ERBB2), KRAS, BRAF, ALK, ROS1, RET, and MET promotes uncontrolled cell proliferation and survival. In contrast, loss or inactivation of tumor suppressor genes including TP53, RB1, PTEN, and BRCA1/2 removes important regulatory mechanisms that normally prevent malignant transformation. These molecular abnormalities act as potential therapeutic targets and form the basis of precision oncology [2,12,13].

Several intracellular signalling pathways play critical roles in cancer development and have become important pharmacological targets. The RAS–RAF–MEK–ERK (MAPK) pathway regulates cell proliferation and differentiation, while the PI3K–AKT–mTOR pathway controls cell growth, metabolism, and survival. Abnormal activation of these pathways is commonly observed in many malignancies and causes uncontrolled tumor growth. Targeted inhibition of these signalling cascades has improved treatment outcomes in selected patient populations carrying actionable genetic modifications [6,13].

Receptor tyrosine kinases are important molecular targets in cancer therapy. EGFR, HER2, VEGFR, PDGFR, KIT, and ALK control cell signalling through phosphorylation. Mutations, gene amplification, or chromosomal rearrangements in these receptors can cause continuous signalling that promotes tumor growth. Tyrosine kinase inhibitors and monoclonal antibodies block these abnormal signals and are widely used to treat several cancers [6,13–15].

Defects in DNA damage repair also play an important role in cancer development. Mutations in BRCA1 and BRCA2 reduce the ability of cancer cells to repair DNA through homologous recombination. This makes them more dependent on other DNA repair pathways. PARP inhibitors target this weakness and can cause cancer cell death through synthetic lethality. They are used in selected patients with ovarian, breast, pancreatic, and prostate cancers with BRCA mutations or related repair defects [4,6].

Predictive biomarkers also play a key role in precision oncology. These measurable molecular features can provide information about treatment response, prognosis, and drug resistance. Common biomarkers include gene mutations, gene amplification, chromosomal translocations, protein overexpression, microsatellite instability (MSI), tumor mutational burden (TMB), and programmed death-ligand 1 (PD-L1) expression. Biomarker testing helps clinicians select suitable treatments for individual patients [4,7,8].

Molecular diagnostic technologies have improved the detection of these biomarkers. Next-generation sequencing (NGS) can examine several cancer-related genes at the same time from small tissue samples. Polymerase chain reaction (PCR), fluorescence in situ hybridization (FISH), immunohistochemistry (IHC), and liquid biopsy are also important diagnostic methods. Liquid biopsy detects circulating tumor DNA (ctDNA) in blood and can assist in monitoring disease progression, identifying resistance mutations, and assessing treatment response [2,4,10].

Pharmacogenomics studies how inherited genetic differences affect drug metabolism, treatment response, and toxicity. Differences in drug-metabolizing enzymes, transport proteins, and drug targets can affect how patients respond to treatment. Pharmacogenomic information can therefore support drug selection, dose adjustment, and medication safety [4,7].

Multi-omics has supplemented the field of precision oncology beyond genomic testing. Transcriptomics, proteomics, metabolomics, and epigenomics provide additional information about tumor biology. Combining these data with clinical information can improve tumor classification, identify new

therapeutic targets, and help predict treatment response. Artificial intelligence and machine learning are also being used to analyse these complex datasets and support personalized treatment decisions [2–4]. A better understanding of cancer biology has changed drug development and clinical practice. The identification of molecular changes, along with advances in biomarker testing and pharmacogenomics, has strengthened the foundation of precision oncology. These developments continue to support new targeted therapies and more customized cancer treatment across different types of cancer. The identification of specific molecular variations has changed cancer treatment by supporting the development of targeted therapies against key cancer-related pathways. Fig 2 provides an overview of the major molecular targets and their corresponding targeted therapeutic strategies used in precision oncology.

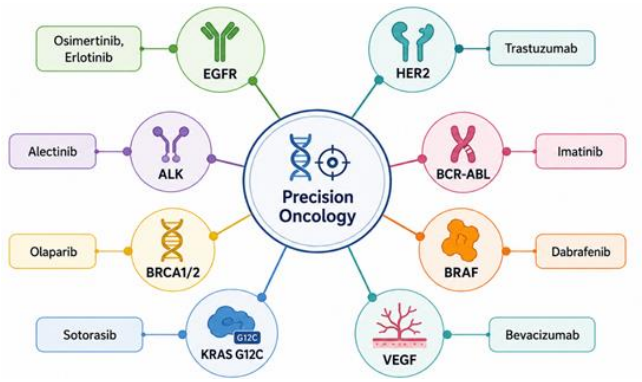

Fig 2 Major Molecular Targets and Targeted Therapeutic Strategies in Precision Oncology

The molecular targets and therapeutic strategies illustrated in Fig 2 form the basis of modern targeted cancer therapy. Understanding these targets is essential for selecting appropriate pharmacological agents, which are discussed in the following section.

The molecular changes associated with different cancers have led to the development of targeted therapies that act on specific proteins involved in tumor growth and progression. Table 1 presents the major molecular targets, representative targeted drugs, associated cancer types, and their main therapeutic effects in precision oncology.

Table 1. Major Molecular Targets and Representative Targeted Therapies Used in Precision Oncology

Molecular target	Associated cancer	Representative targeted drug(s)	Main therapeutic effect
EGFR	Non-small cell lung cancer	Osimertinib, Erlotinib, Gefitinib	Inhibits tumor cell growth by blocking EGFR signalling
HER2	Breast cancer, Gastric cancer	Trastuzumab, Pertuzumab	Reduces HER2-mediated tumor proliferation
BCR-ABL	Chronic myeloid leukaemia	Imatinib, Dasatinib	Blocks abnormal BCR-ABL tyrosine kinase activity
ALK	Non-small cell lung cancer	Alectinib, Crizotinib	Inhibits ALK-driven tumor growth
BRAF	Melanoma	Vemurafenib, Dabrafenib	Suppresses MAPK pathway signalling
KRAS G12C	Non-small cell lung cancer	Sotorasib, Adagrasib	Selectively inhibits mutant KRAS protein
BRCA1/BRCA2	Breast, Ovarian, Prostate cancers	Olaparib, Niraparib	Induces cancer cell death by inhibiting DNA repair
VEGF	Colorectal, Renal and other solid tumors	Bevacizumab	Inhibits angiogenesis and reduces tumor blood supply
PI3K	Breast cancer	Alpelisib	Inhibits the PI3K/AKT signalling pathway
RET	Thyroid cancer, non-small cell lung cancer	Selpercatinib	Blocks RET-mediated signalling

The molecular targets summarized in Table 1 provide the basis for the development of modern targeted anticancer drugs. Understanding how these drugs act on specific targets is important for assessing their role in precision oncology. The following section describes the major classes of targeted anticancer agents, their mechanisms of action, pharmacological properties, and clinical applications.

Pharmacology of Targeted Anticancer Agents

Targeted anticancer agents have become an important component of precision oncology because they selectively inhibit molecular abnormalities responsible for tumor growth and progression. In conventional chemotherapy both normal and rapidly dividing cancer cells are affected but targeted therapies only act on specific proteins or signalling pathways that contribute to cancer development. This selective mechanism improves therapeutic precision and has led to better clinical outcomes in several malignancies while reducing unnecessary toxicity to healthy tissues [6,8].

Targeted therapies are broadly classified into small-molecule inhibitors and monoclonal antibodies. Small-molecule inhibitors are usually administered orally and penetrate cells to inhibit intracellular enzymes, particularly protein kinases involved in cancer signalling pathways. Monoclonal antibodies are administered intravenously and bind to specific cell surface receptors or circulating ligands, preventing the activation of signalling pathways or enhancing immune-mediated destruction of cancer cells. Both classes are crucial in the treatment of several solid tumors and haematological malignancies [6,13]. Tyrosine kinase inhibitors (TKIs) represent the largest class of targeted anticancer drugs. These agents inhibit abnormal kinase activity by blocking ATP binding to tyrosine kinase enzymes. they also act by interrupting signalling pathways that promote cell proliferation and survival. Imatinib was the first successful TKI and revolutionized the treatment of chronic myeloid leukaemia by specifically inhibiting the BCR-ABL tyrosine kinase [5]. Since then, several TKIs such as osimertinib, erlotinib, gefitinib, alectinib, and crizotinib have demonstrated significant clinical benefit in patients with EGFR-mutated or ALK-positive non-small cell lung cancer and other malignancies [13–15].

Monoclonal antibodies are designed to recognize specific antigens expressed on tumor cells or molecules involved in tumor progression. Trastuzumab targets the HER2 receptor and has significantly improved survival in patients with HER2-positive breast and gastric cancers. Bevacizumab inhibits vascular endothelial growth factor (VEGF), thereby reducing tumor angiogenesis. cetuximab blocks epidermal growth factor receptor (EGFR) signalling in selected patients with colorectal and head and neck cancers. These agents are frequently combined with chemotherapy or other targeted therapies to enhance treatment efficacy [6,15].

Antibody-drug conjugates (ADCs) are an important development in targeted drug delivery. They combine monoclonal antibodies with highly potent cytotoxic drugs using a stable chemical linker. After the ADC binds to a specific antigen, it enters the cancer cell and releases the cytotoxic drug, which causes cell death. This targeted action can improve treatment selectivity and reduce toxicity to normal tissues. Trastuzumab, deruxtecan and Sacituzumab, govitecan are examples of ADCs that have displayed promising clinical outcomes in breast cancer and other solid tumors [19].

Poly (ADP-ribose) polymerase (PARP) inhibitors act mainly on cancer cells with defects in homologous recombination DNA repair, especially those with BRCA1 or BRCA2 mutations. These drugs block PARP enzymes and reduce DNA repair, causing DNA damage to build up in cancer cells. This can lead to selective cancer cell death through synthetic lethality. Olaparib, niraparib, rucaparib, and talazoparib are used in selected patients with ovarian, breast, pancreatic, and prostate cancers [4].

Cyclin-dependent kinase (CDK) 4/6 inhibitors have improved the treatment of hormone receptor-positive, HER2-negative advanced breast cancer. Palbociclib, ribociclib, and abemaciclib block cell-cycle progression by preventing phosphorylation of the retinoblastoma protein, which causes cell-cycle arrest in the G1 phase. When these drugs are used along with endocrine therapy, they can prolong progression-free survival and delay disease progression [20].

Targeting the MAPK signalling pathway is an effective treatment approach for tumors with BRAF mutations. BRAF inhibitors such as vemurafenib and dabrafenib are generally combined with MEK inhibitors to improve treatment response and reduce the development of drug resistance. This combination is a standard treatment option for advanced melanoma and for selected patients with colorectal cancer carrying BRAF mutations [21]. Other targeted therapies are also expanding treatment options in precision oncology. PI3K inhibitors are used in selected patients with PIK3CA-mutated breast cancer. KRAS G12C inhibitors provide new treatment options for patients with specific types of non-small cell lung cancer. Selective inhibitors targeting RET and MET alterations have also exhibited positive clinical results, supporting the continuous growth of biomarker-guided cancer therapy [13,16].

Targeted therapies can cause adverse effects that require careful monitoring. The type and severity of these effects depend on the molecular target and drug class. Common adverse effects like skin rashes, diarrhoea, hypertension, hepatotoxicity, cardiotoxicity, fatigue, and blood-related abnormalities may occur. Early recognition and proper management of these toxicities can help patients continue treatment and improve therapeutic outcomes [4,6].

Gradual development of targeted anticancer agents has transformed the pharmacological management of cancer and strengthened the role of precision oncology in clinical practice. Advances in molecular pharmacology, biomarker-guided therapy, and drug development continue to expand treatment options. This paves a path for providing more personalized and effective care for patients with different types of cancer. The pharmacological properties of the major classes of targeted anticancer drugs vary according to their molecular targets, mechanisms of action, and clinical applications. Table 2 summarizes the important characteristics of commonly used targeted therapies in precision oncology.

Table 2. Pharmacological Characteristics of Major Classes of Targeted Anticancer Drugs

Drug class	Representative drugs	Primary target	Common clinical use	Common adverse effects
Tyrosine kinase inhibitors (TKIs)	Imatinib, Osimertinib, Alectinib	BCR-ABL, EGFR, ALK	Chronic myeloid leukaemia, non-small cell lung cancer	Skin rashes, diarrhoea, hepatotoxicity
Monoclonal antibodies	Trastuzumab, Bevacizumab, Cetuximab	HER2, VEGF, EGFR	Breast, gastric, colorectal cancers	Infusion reactions, cardiotoxicity, hypertension
PARP inhibitors	Olaparib, Niraparib	PARP enzymes	BRCA-mutated breast and ovarian cancers	Anaemia, nausea, fatigue
CDK4/6 inhibitors	Palbociclib, Ribociclib, Abemaciclib	CDK4/6	HR-positive, HER2-negative breast cancer	Neutropenia, diarrhoea, fatigue
BRAF inhibitors	Vemurafenib, Dabrafenib	BRAF V600 mutation	Melanoma	Skin toxicity, arthralgia, photosensitivity
PI3K inhibitors	Alpelisib	PI3K	PIK3CA-mutated breast cancer	Hyperglycaemia, rash, diarrhoea

The pharmacological diversity of targeted anticancer agents provides health care providers with multiple treatment options for different molecular subtypes of cancer. Appropriate drug selection depends on accurate biomarker identification and a complete molecular testing. The following section discusses the importance of biomarkers and companion diagnostics in guiding personalized treatment decisions in precision oncology.

Biomarkers and Companion Diagnostics in Precision Oncology

Biomarkers play an important role in precision oncology because they help identify patients who may benefit from targeted therapies. A biomarker is a measurable biological feature that provides information about the presence of a disease, response to treatment, or disease prognosis. In cancer

care, biomarkers support personalized treatment by linking specific molecular changes to suitable targeted drugs. This approach can improve treatment selection and therapeutic outcomes [2,8].

Predictive biomarkers help clinicians choose the right targeted treatment for each patient. For example, EGFR mutations can show whether a patient with non-small cell lung cancer may respond to EGFR tyrosine kinase inhibitors. HER2 overexpression can identify patients who may benefit from HER2-targeted drugs. BRCA1/2 mutations can help select patients for PARP inhibitor treatment. Testing for the BCR-ABL fusion gene is also important before using imatinib and other BCR-ABL inhibitors [4,5,15]. Companion diagnostics are laboratory tests used to identify important biomarkers before treatment starts. They help health care professionals choose the right treatment by confirming specific molecular changes in cancer cells. Common methods like polymerase chain reaction (PCR), fluorescence in situ hybridization (FISH), immunohistochemistry (IHC), and next-generation sequencing (NGS) may be selected. These tests support personalized treatment planning and assist in avoiding treatments that may not work [10,11].

Among the available diagnostic methods, next-generation sequencing (NGS) is an important tool in precision oncology because it can test many cancer-related genes at the same time. Compared with testing one gene at a time, NGS can provide detailed genetic information using a small amount of tumor tissue. This aids in identifying mutations that can be treated with targeted drugs and supports in treatment selection across different types of cancer [2,10].

Liquid biopsy is a useful alternative for conventional tissue biopsy in some situations. It uses blood samples to detect circulating tumor DNA and other substances released by cancer cells. It can assist in detecting genetic changes, monitor treatment response, identify resistance mutations, and detect cancer recurrence. Tissue biopsy remains the standard method for initial diagnosis, while liquid biopsy is increasingly used for patient follow-up and disease monitoring [18].

Pharmacogenomics also contributes in personalized cancer treatment by evaluating inherited genetic variations that influence drug metabolism, efficacy, and toxicity. Variations in genes encoding drug-metabolizing enzymes, transport proteins, and drug targets can affect individual responses to anticancer therapy. Integrating pharmacogenomic information with clinical decision-making supports in safer drug selection, dose optimization, and improved patient care [4,7]. The growing number of useful biomarkers has increased the need for clear systems to interpret molecular findings. The ESMO Scale for Clinical Actionability of Molecular Targets (ESCAT) helps classify genetic changes based on their clinical importance and the evidence supporting targeted treatment. These systems benefit clinicians in identifying the molecular changes that are most useful for treatment decisions [22].

Precision oncology depends on the proper use of biomarker testing, companion diagnostics, pharmacogenomics, and clinical evaluation. As molecular diagnostic methods continue to improve, biomarker-guided treatment can help select suitable therapies, improve treatment outcomes, and support the development of personalized cancer care. Validated companion diagnostic tests help in recognizing predictive biomarkers and select suitable targeted therapies in precision oncology. Table 3 summarizes the major biomarkers, common diagnostic methods, and representative targeted therapies used in clinical practice.

Biomarker-guided treatment has improved the selection of targeted therapies and supports personalized cancer care. Based on this approach, precision oncology has changed the treatment of many cancers by matching specific molecular changes with suitable targeted therapies.

The successful implementation of precision oncology depends on the systematic integration of molecular diagnostics, biomarker identification, and targeted therapy selection. Fig 3 illustrates the workflow of biomarker-guided precision oncology, from cancer diagnosis to treatment monitoring and therapeutic decision-making.

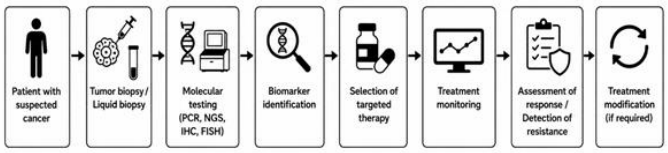
Patient with suspected cancer
Tumor biopsy / Liquid biopsy
Molecular testing (PCR, NGS, IHC, FISH)
Biomarker identification
Selection of targeted therapy
Treatment monitoring
Assessment of response / Detection of resistance
Treatment modification (if required)

Fig 3 Workflow of Biomarker-Guided Precision Oncology

The biomarker-guided workflow shown in Fig 3 highlights the importance of integrating molecular testing with clinical decision-making to optimize targeted therapy. This systematic process has improved personalized treatment and forms the basis for the clinical applications of targeted therapies discussed in the following section.

3.5 Clinical Applications of Targeted Therapy in Major Cancers

Targeted therapies have changed the management of many cancers by providing treatments based on the molecular characteristics of individual tumors. The selection of these therapies depends on biomarker testing, which determines whether a patient is suitable for a specific drug. This personalized treatment strategy has improved clinical outcomes, reduced unnecessary exposure to ineffective therapies, and strengthened the role of precision oncology in routine cancer care [2,8].

Non-small cell lung cancer (NSCLC) is a major example of precision oncology in clinical practice. Molecular testing for changes in EGFR, ALK, ROS1, KRAS, RET, and MET is now a standard part of patient evaluation. Patients with EGFR mutations are commonly treated with EGFR tyrosine kinase inhibitors such as osimertinib and ALK-positive tumors are treated with ALK inhibitors such as alectinib. KRAS G12C inhibitors have also added new treatment options for selected patients with advanced NSCLC [14,16,23].

In breast cancer, targeted therapy has improved the treatment of HER2-positive disease. Monoclonal antibodies such as trastuzumab and pertuzumab are commonly given with

chemotherapy to improve survival. For hormone receptor-positive, HER2-negative breast cancer, CDK4/6 inhibitors can be used with endocrine therapy. PARP inhibitors are also used in selected patients with BRCA1 or BRCA2 mutations [15,20,23].

Targeted therapy is also vital part of colorectal cancer treatment. Patients are tested for RAS and BRAF mutations before treatment selection. Anti-EGFR monoclonal antibodies such as cetuximab are used in patients without RAS mutations. For patients with BRAF V600E-mutated disease, BRAF-targeted combination therapy provides another treatment option [21,23]. Patients with ovarian, breast, pancreatic, and prostate cancers carrying BRCA1/2 mutations have benefited from the introduction of PARP inhibitors. These agents have improved progression-free survival in selected patients by targeting defects in DNA repair pathways. Genetic testing has therefore become an important part of treatment planning in these cancers [4,23]. The growing availability of molecular testing has expanded the use of targeted therapies in several other malignant conditions like thyroid cancer, gastric cancer, melanoma, and renal cell carcinoma. Continuous research is identifying additional molecular targets and supporting the development of new therapies that further strengthen the role of precision oncology in cancer management [12,23]. Successful treatment depends on accurate biomarker testing, appropriate patient selection, and regular monitoring of treatment response and adverse effects. Multidisciplinary collaboration among oncologists, pathologists, pharmacists, and molecular diagnostic laboratories is essential for the effective use of precision oncology in clinical practice [10,11,24].

The increasing use of targeted therapies in different types of cancers determines the importance of integrating molecular diagnostics with pharmacological treatment. As new biomarkers and targeted drugs emerge, precision oncology may improve personalized cancer care and clinical outcomes gradually. The effectiveness of targeted therapies may decrease over time because of the development of resistance through various molecular and cellular mechanisms. Understanding these mechanisms is important for selecting appropriate treatment strategies and improving long-term clinical outcomes. Table 4 summarizes the common mechanisms of resistance to targeted therapies and the potential strategies used to overcome them.

Table 3. Major Predictive Biomarkers and Companion Diagnostic Tests Used in Precision Oncology

Biomarker	Associated cancer	Companion diagnostic test	Representative targeted therapy	Clinical significance
EGFR mutation	Non-small cell lung cancer	PCR, NGS	Osimertinib, Erlotinib	Predicts response to EGFR tyrosine kinase inhibitors
HER2 overexpression/amplification	Breast cancer, Gastric cancer	IHC, FISH	Trastuzumab, Pertuzumab	Identifies patients eligible for HER2-targeted therapy
BCR-ABL fusion	Chronic myeloid leukaemia	RT-PCR, FISH	Imatinib, Dasatinib	Confirms diagnosis and guides TKI therapy
ALK rearrangement	Non-small cell lung cancer	FISH, IHC, NGS	Alectinib, Crizotinib	Predicts response to ALK inhibitors
BRAF V600 mutation	Melanoma, Colorectal cancer	PCR, NGS	Dabrafenib, Vemurafenib	Identifies candidates for BRAF-targeted therapy
BRCA1/BRCA2 mutation	Breast, Ovarian, Prostate, Pancreatic cancers	NGS	Olaparib, Niraparib	Predicts response to PARP inhibitors
PIK3CA mutation	Breast cancer	PCR, NGS	Alpelisib	Supports selection of PI3K inhibitor therapy
KRAS G12C mutation	Non-small cell lung cancer	PCR, NGS	Sotorasib, Adagrasib	Identifies patients suitable for KRAS-targeted therapy
RET	Thyroid cancer, non-small cell lung cancer	NGS, FISH	Selpercatinib, Pralsetinib	Identifies patients who benefit from selective RET inhibitors

Table 4. Common Mechanisms of Resistance to Targeted Therapy and Strategies to Overcome Resistance

Targeted therapy	Common mechanism of resistance	Clinical impact	Strategy to overcome resistance
EGFR inhibitors	Secondary EGFR mutations, activation of alternative pathways	Reduced treatment response	Next-generation EGFR inhibitors, combination therapy
ALK inhibitors	Secondary ALK mutations	Disease progression	Later-generation ALK inhibitors
HER2-targeted therapy	HER2 alterations, activation of bypass pathways	Reduced drug sensitivity	Combination HER2-targeted therapy
BRAF inhibitors	Reactivation of the MAPK pathway	Tumor recurrence	Combined BRAF and MEK inhibition
PARP inhibitors	Restoration of homologous recombination repair	Reduced efficacy	Combination therapy and development of new PARP inhibitors
CDK4/6 inhibitors	Alterations in cell-cycle regulatory proteins	Disease progression	Alternative targeted agents and combination therapy

The development of resistance is still a major challenge in precision oncology despite the remarkable success of targeted therapies. Ongoing research is focused on developing new drugs, combination treatment strategies, and innovative technologies to improve treatment response and delay resistance. These recent advances are discussed in the following section.

3.6 Mechanisms of Resistance to Targeted Therapy

Targeted therapies have improved the treatment of many cancers by blocking molecular pathways involved in tumor growth. However, many patients eventually develop drug resistance, which can cause disease progression and reduce treatment effectiveness. Resistance may be present before treatment starts (primary resistance) or may develop after an

initial response (acquired resistance). Understanding these mechanisms is important for improving treatment outcomes and developing new therapeutic strategies [17].

One common mechanism of resistance is the development of secondary mutations in the target protein. These mutations can change the structure of the target and reduce drug binding, which lowers treatment effectiveness. A well-known example is the development of secondary mutations in the epidermal growth factor receptor (EGFR), which can reduce the effect of EGFR tyrosine kinase inhibitors in patients with non-small cell lung cancer [14,25].

Cancer cells can also activate alternative signalling pathways to maintain their growth and survival when the main target is blocked. This process is known as bypass signalling. It allows cancer cells to continue growing even when one signalling pathway is inhibited. Activation of MET, HER2, and other receptor tyrosine kinases has been reported as a resistance mechanism in several cancers [13,17].

Another important factor is tumor heterogeneity, which means that different groups of cancer cells can exist within the same tumor. Targeted therapy may destroy sensitive cancer cells, while resistant cells can survive and increase over time. This can lead to disease recurrence and make long-term treatment more difficult [17,23].

Changes in the tumor microenvironment can also affect treatment response. Cancer cells interact with immune cells, stromal cells, and surrounding tissues, and these interactions can support tumor survival and reduce the effect of targeted therapies. These interactions are being studied as possible targets for new treatments [24]. Drug resistance can also result from pharmacological changes, such as reduced drug concentration inside cancer cells, changes in drug metabolism, or increased drug efflux. These changes reduce the amount of active drug reaching its target and can contribute to treatment failure [4,17].

Several strategies are used to manage drug resistance. Next-generation targeted therapies have improved treatment options for patients who develop resistance to earlier drugs. For example, newer EGFR and ALK inhibitors have been developed to overcome specific resistance mutations. Combination therapy can also target more than one signalling pathway at the same time and reduce the chance of bypass pathway activation. Regular molecular monitoring through tissue biopsy or liquid biopsy can detect new resistance mechanisms and support timely changes in treatment [18,24,25].

Persistent research into the molecular basis of drug resistance is supporting the development of better treatment strategies. development in molecular diagnostics, combination therapies, and new targeted drugs may delay resistance, improve treatment response, and strengthen the role of precision oncology in cancer management.

3.7 Emerging Therapeutic Strategies and Future Directions

Continuous progress in cancer biology, molecular diagnostics, and pharmacology is expanding the scope of precision oncology. New treatment approaches are also being developed to improve treatment response, reduce drug resistance, and provide more personalized care for patients with different types of cancer. These developments are expected to strengthen the role of targeted therapy in future cancer treatment [24].

One major area of progress is the expansion of next-generation targeted therapies with greater selectivity and lower toxicity. These newer drugs are designed to overcome resistance to earlier treatments by targeting newly discovered molecular changes or blocking multiple signalling pathways involved in tumor growth. Ongoing drug development is also increasing the number of molecular targets available for cancer treatment [13,17].

Antibody-drug conjugates (ADCs) are another vital area for progress in targeted drug delivery. They combine monoclonal antibodies with potent cytotoxic drugs to deliver treatment directly to cancer cells while reducing damage to normal tissues. Several newer ADCs have demonstrated encouraging results in breast, lung, and other solid tumors. Ongoing clinical studies may further expand their use in cancer treatment [19].

Combination therapy is also an important strategy in precision oncology. Targeted drugs may be used together or combined with chemotherapy, endocrine therapy, or immunotherapy. Using different treatments together can improve treatment response, delay drug resistance, and block several cancer-related pathways. This approach is now used in the treatment of several cancers [17,21].

Advances in liquid biopsy and next-generation sequencing have improved the detection of genetic changes that can guide treatment and resistance mutations. These methods can support diagnosis, treatment selection, and regular monitoring of disease using minimally invasive techniques. Their increasing use may improve customized treatment planning and support timely changes in therapy when resistance develops [18,24].

The use of artificial intelligence (AI) and machine learning is another developing area in precision oncology. AI-based systems can analyse genomic data, medical images, and clinical records to support biomarker detection, assess treatment response, and assist in treatment selection. These technologies are still developing but may improve clinical decision-making and patient care [3].

Multi-omics technologies provide another important direction for precision oncology. They combine genomic, transcriptomic, proteomic, and metabolomic data to provide a broader view of tumor biology. This approach can support biomarker discovery, the identification of new therapeutic targets, and better assessment of treatment response [2,24].

Future progress in targeted cancer therapy will require continued collaboration among researchers, clinicians, pharmacists, and regulatory agencies. Rapid growth in molecular diagnostics, drug development, computational technologies, and personalized treatment approaches can improve clinical outcomes and expand the benefits of precision

oncology to more patients. Recent developments in molecular diagnostics, targeted drug development, and computational technologies expand the scope of precision oncology. Fig 4 summarizes the major emerging therapeutic strategies and future directions that may further improve personalized cancer treatment.

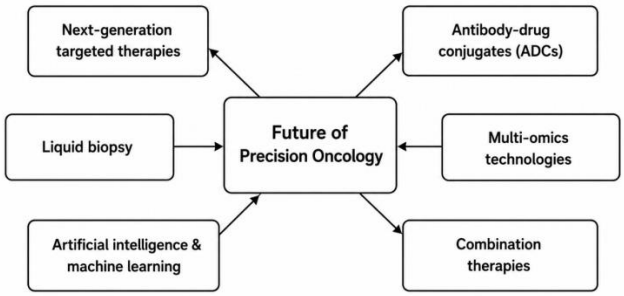

Fig 4 Emerging Therapeutic Strategies and Future Directions in Precision Oncology

The emerging strategies presented in Fig 4 highlight the evolution of targeted cancer therapy and their potential to improve patient care. These developments also highlight the opportunities and challenges associated with implementing precision oncology in clinical practice, which are discussed in the following section.

Discussion

Targeted cancer therapy has changed modern oncology by moving treatment from conventional chemotherapy toward drugs that act on specific molecular changes in cancer cells. Advances in molecular biology, pharmacogenomics, and precision oncology have improved the detection of useful biomarkers and supported the development of selective targeted drugs. These developments have improved treatment response, extended survival, and enhanced quality of life for patients with several types of cancer [8,31]. One of the main strengths of targeted therapy is its ability to provide personalized treatment based on the molecular profile of each tumor. Biomarker-guided drug selection can improve treatment effectiveness by selecting patients who are suitable for specific therapies and reducing unnecessary use of ineffective treatments. The use of companion diagnostics with targeted therapy has therefore become an essential part of precision oncology and continues to support treatment decisions in different types of cancer [24,30]. Targeted therapy has also several important limitations. Drug resistance remains one of the major barriers to long-term treatment success and can result in disease progression after an initial period of clinical benefit. Tumor heterogeneity, secondary genetic changes, and activation of alternative signalling pathways can reduce the effectiveness of many targeted drugs. Continuous molecular monitoring and the development of next-generation targeted therapies are important strategies for managing these challenges [25,33]. Another challenge is the implementation of precision oncology in routine clinical practice. Comprehensive molecular testing, next-generation sequencing, and companion diagnostic technologies require specialized infrastructure, trained personnel, and financial resources. Limited access to these facilities may affect the availability of personalized treatment in many healthcare settings. Continuous investment in molecular diagnostic services and access to targeted therapies will be important for expanding the clinical benefits of precision oncology [26].

The growing amount of genomic and clinical data has increased the need for bioinformatics and clinical decision-support systems. Knowledge bases and computer-based tools can assist clinical professionals in understanding complex molecular information, finding genetic changes that can be targeted, and selecting treatments based on available evidence. These technologies will become more important as genomic profiling becomes a regular part of cancer care [27,32].

Pharmacists play an important role in the use of targeted cancer therapy. Their work goes beyond dispensing medicines and includes patient counselling, monitoring adverse drug reactions, checking for drug interactions, supporting medication adherence, and working with multidisciplinary oncology teams. Pharmacists can also assist with the interpretation of pharmacogenomic information and support the safe and effective use of targeted therapies throughout treatment [4,7].

Future progress in targeted cancer therapy will require continuous collaboration among clinicians, pharmacists, molecular biologists, bioinformaticians, and regulatory organizations. Advances in artificial intelligence, multi-omics technologies, liquid biopsy, and real-world clinical data can improve biomarker discovery, support treatment selection, and contribute to the development of more effective targeted therapies. Constant research and better access to precision oncology services can further improve patient outcomes and strengthen the role of targeted therapy in modern cancer care [28,29]. Overall, targeted cancer therapy has established a strong foundation for personalized cancer treatment. Recent and gradual advancement in pharmacology, molecular diagnostics, and precision medicine are expected to improve therapeutic outcomes, reduce treatment-related toxicity, and expand the application of precision oncology across a broader range of malignancies [29,31].

Conclusion

Targeted cancer therapy has transformed the management of cancer by shifting treatment from a generalized approach to one based on the molecular characteristics of individual tumors. The identification of actionable biomarkers, advances in molecular diagnostics, and the development of targeted drugs have improved treatment precision and clinical outcomes in several malignancies. These advances established precision oncology as an important part of modern cancer care. The use of pharmacogenomics, companion diagnostics, and biomarker-guided treatment has improved patient selection and supported the appropriate use of targeted therapies. Small-molecule inhibitors, monoclonal antibodies, antibody-drug conjugates, and other targeted drugs have increased treatment options for many cancers and reduced the use of ineffective therapies. Still, drug resistance, tumor heterogeneity, limited access to molecular testing, and the high cost of targeted therapies remain important challenges for their wider use in clinical practice. Recent advancement in next-generation sequencing, liquid

biopsy, artificial intelligence, and multi-omics technologies are strengthening the practice of precision oncology by improving diagnosis, treatment selection, and disease monitoring. Continuous research into new molecular targets and innovative therapeutic strategies is expected to improve the effectiveness and safety of targeted cancer therapies.

The successful implementation of precision oncology requires collaboration among clinicians, pharmacists, researchers, molecular biologists, and diagnostic laboratories. Pharmacists play an important role in optimizing targeted therapy through medication management, patient counselling, monitoring for adverse effects, and promoting adherence to treatment. Their contribution is essential for achieving safe and effective use of targeted anticancer agents.

In conclusion, targeted cancer therapy continues to reshape the field of oncology by providing more personalized and effective treatment strategies. Ongoing developments in pharmacology, molecular diagnostics, and precision medicine are expected to expand the availability of targeted therapies and improve the quality of cancer care. Continuous innovation and multidisciplinary collaboration will further strengthen the role of precision oncology in achieving better outcomes for patients with cancer.

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AI Tool Declaration

The authors declare that no AI and related tools are used to write the scientific content of this manuscript.

Conflict of Interests

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Data Availability

Data will be available on request

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